

Efficient community-based control strategies in adaptive networks

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Most researches on adaptive networks mainly concentrate on the properties of steady state, but neglect transient dynamics. In this study, we pay attention to the emergence of community structures in transient process and the effects of community-based control strategies on epidemic spreading. First, by normalizing modularity Q , we investigate the evolution of community structures during the transient process, and find that very strong community structures are induced by rewiring mechanism in the early stage of epidemic spreading, which remarkably delays the outbreaks of epidemic. Then we study the effects of control strategies started from different stages on the prevalence. Both immunization and quarantine strategies indicate that it is not “the earlier, the better” for the implementing of control measures. And the optimal control effect is obtained if control measures can be efficiently implemented in the period of strong community structure. For immunization strategy, immunizing the S nodes on SI links and immunizing S nodes randomly have similar control effects. Yet for quarantine strategy, quarantining the I nodes on SI links can yield far better effects than quarantining I nodes randomly. More significantly, community-based quarantine strategy plays more efficient performance than community-based immunization strategy. This study may shed new lights on the forecast and the prevention of epidemic among human population.

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In various real-world systems, the structures are constantly changing with the states of the network and vice versa [1]. For instance, frequent traffic congestions on a road may lead to the building of new roads, while the new roads will influence the traffic flow on some roads [2]. These phenomena are characterized by the existence of a feedback loop between the dynamics in networks and the dynamics of networks, and the networks with such a feedback loop are called coevolutionary or adaptive network [2, 3]. Until recently, a variety of adaptive networks have been studied, such as biological networks [4], chemical networks [5], ecological systems [6, 7] and technological networks [8]. And many interesting phenomena are found, including the robust self-organization phenomenon in biological nervous systems [9, 10], the promotion cooperation in evolutionary game [11, 12], the emergence of community structure in opinion spreading [13], etc.

From epidemiological viewpoint, when an infectious disease appears in a population, human self-protection behaviors can significantly change the predicted course of epidemics [14, 15]. In the extreme, susceptible people may break their contacts with infected partners. This will significantly alter the structure of the contact network, thus influencing the pathway of epidemic spreading. Gross *et al.* first studied the dynamics of susceptible-infected-susceptible(SIS) model in adaptive networks, and found that different epidemic transmis-

sion rates and rewiring rates can lead to fascinating phenomena, like bistability and circles [16]. Shaw and Schwartz [17] considered a susceptible-infected-recovery-susceptible (SIRS) model in adaptive networks, and came into similar phenomena. Subsequently, Marceau *et al.* developed a more precise analytical method for the adaptive networks in the framework of the model of Gross [18]. In addition, quite a few studies showed that adaptive networks can effectively change the dynamics of epidemic [19, 20] and contact switching is an effective control strategy for epidemic outbreaks [21–23].

The previous studies of epidemic spreading in adaptive networks mainly focus on steady state while ignoring transient dynamics. However, the understanding of transient dynamics would help to put forward efficient and timely control strategy. In this paper, we look into the adaptive SIS model of Gross *et al.* [16] again, and find that very strong community structures are induced by rewiring mechanism in the early stage of epidemic spreading. Then two community-based control strategies are proposed, and a counter-intuitive conclusion is discovered: it is not “the earlier, the better” for the implementing of control measures.

The model considers SIS dynamics in a random network with fixed N nodes and K undirected links [16], where a node may be in S or I state. For every SI link, I node infects S node with fixed probability p per time step. And I node recovers from the disease with probability r , becoming susceptible again. Meanwhile, with probability w for every SI link, S node rewires the link to a randomly selected S node. Multiple connections and self-connections are excluded. Results presented in the following are for $N = 10^4$ and $K = 10^5$.

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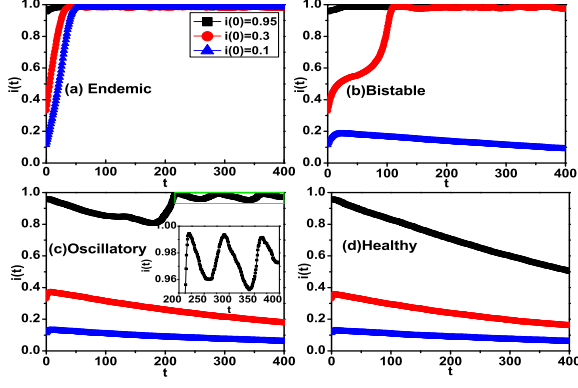


FIG. 1: (Color online) The density of infected $i(t)$ (i. e., the prevalence) versus time t for different initial densities of infected $i(0)$, where “squares”, “circles” and “triangles” denote the results of $i(0) = 0.95, 0.3, 0.1$, respectively. Four figures represent different phases: (a) Endemic state, $w = 0.1$; (b) Bistable state, $w = 0.3$; (c) Oscillatory state, $w = 0.6$; (d) Healthy state, $w = 0.7$. Here $p = 0.008, r = 0.002$. Inset: the magnification of the black line labelled green region.

For different values of w and p , the system can be divided into four different phases [16]: endemic state, bistable state, oscillatory state, and healthy state (see Fig. 1). Note, in bistable state and oscillatory state, different initial densities of infected $i(0)$ can yield completely different results. In Fig. 1(b), for $i(0) = 0.1$, large rewiring rate w can effectively isolate I nodes and prevent S nodes from infection, leading to the dying out of epidemic. For $i(0) = 0.3$, the rewiring behavior can lower the outbreak velocity at the early stage, but can not prevent the outbreak of epidemic. For $i(0) = 0.95$, the impact of rewiring behavior is negligible due to the huge number of I nodes. When w increases to 0.6, there is an oscillatory state for large $i(0) = 0.95$ in Fig. 1(c). Though large rewiring rate w can prevent the prevalence to some extent, the gradually increasing S nodes will form a giant and tight cluster with the smaller threshold, which causes re-outbreak in S cluster. This process will repeat so many times, and an oscillatory phenomenon appears.

From above analysis, we know that the rewiring behavior can lead to two separated but internally compact clusters. Naturally, we can define the community structure of the adaptive network according to the different states of nodes [24]. Strong community structure implies that both S and I nodes are more likely to connect to nodes with the same state and the number of SI links is relatively small, while weak community structure indicates that S and I nodes are mixed more fully in the network. Network modularity Q , a popular evaluating indicator in measuring community structure [25], is de-

fined as

$$Q = \sum_{s=1}^C \left[\frac{l_s}{L} - \left(\frac{d_s}{2L} \right)^2 \right], \quad (1)$$

where l_s and d_s represent the number of intra-links and the sum of degrees of the nodes in community s respectively, L denotes the number of links in the network, and C is the number of communities. Here $0 \leq Q \leq 1$, the larger Q is, the stronger community structure is.

Fig. 2 shows the time evolution of $Q(t)$ in different phases. One can find that, sometimes, the indicator $Q(t)$ cannot well characterize the community strength of the network. For example, when the system is in healthy state, most of SI links can be rapidly broken by the large rewiring rate w , which may bring about the complete separation of S and I clusters. Hence the community structure in healthy state should be more obvious than in the other states. Yet from Fig. 2(d), one can find that $Q(t)$ is very low, especially for large time steps. It should be noted that such difference comes from the shortcomings of modularity Q . Specifically, for $C = 2$, Eq. (1) is expanded as

$$Q = \sum_{s=1}^2 \left[\frac{l_s}{L} - \left(\frac{d_s}{2L} \right)^2 \right] = 1 - \frac{l_{12}}{L} - \left(\frac{d_1}{2L} \right)^2 - \left(\frac{d_2}{2L} \right)^2, \quad (2)$$

where l_{12} represents the amount of inter-links between community 1 and 2. When the network is connected randomly, $l_{12} = (d_1 d_2) / 2L$, thus $Q = 0$; When $l_{12} = 0$ and $l_1 = l_2$, Q reaches the maximum, i. e., $Q = 0.5$. Therefore Q can only range from 0 to 0.5. Moreover, the large difference between intra-link number of community 1 and 2, e. g., $d_1 \gg d_2$, also induces small Q even when l_{12} is small. For instance, community A(B) is a complete graph with 50(10) nodes and there is only one link between the two communities. The modularity of such a community network is $Q = 1 - 1/1271 - (2451/2542)^2 - (91/2542)^2 \approx 0.068$.

Above all, Q is not an accurate index to characterize the community structure of the network with two communities. To address this shortcoming, like Ref. [26], the normalized Q_n is defined as

$$Q_n = \frac{Q - Q_{rand}}{Q_{max} - Q_{rand}}, \quad (3)$$

where Q_{rand} corresponds to random network with the same degree sequence, and Q_{max} is the modularity of the network without inter-community links, i. e., $l_{12} = 0$. After this standardization, Q_n can range from 0 to 1.

The normalized Q_n , in comparison to $Q(t)$, can reflect the community structure in the adaptive network more accurately. In endemic state, as shown in Fig. 2 (a), though small rewiring rate w can't prevent the outbreak of epidemic, it can also induce a certain degree of community structure. In bistable state, Fig. 2 (b) demonstrates that $w = 0.3$ can give rise to the form of large and tight S cluster in the early stage ($t \leq 50$). And

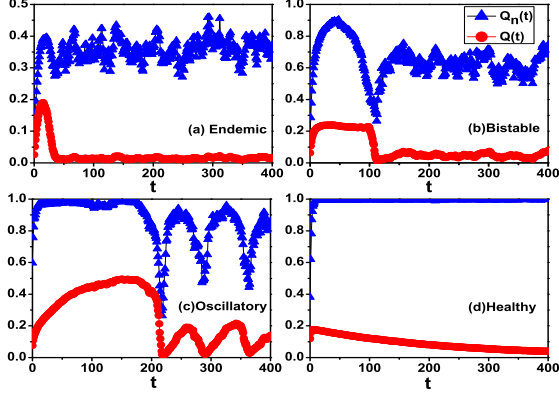


FIG. 2: (Color online) The time evolution of $Q(t)$ (“circles”) and $Q_n(t)$ (“triangles”) under different conditions. (a) $i(0) = 0.3, w = 0.1$; (b) $i(0) = 0.3, w = 0.3$; (c) $i(0) = 0.95, w = 0.6$; (d) $i(0) = 0.3, w = 0.7$. When $i(0)$ is small, oscillatory state is the same as the healthy state; Oscillation occurs only when $i(0)$ is large. So we choose $i(0) = 0.95$ in oscillatory state. Here $p = 0.008, r = 0.002$.

subsequent re-outbreak in S cluster ($50 < t \leq 110$) results in the weakening of community strength. When $t > 110$, the community strength of the network will keep a stable value. In oscillatory state, $w = 0.6$ can isolate I nodes quickly, hence the community strength becomes large rapidly. But with S cluster becomes larger and tighter, the epidemic threshold becomes smaller. Finally, epidemic prevails in S cluster at some point, such as $t \approx 200$ in Fig. 2(c), resulting in the weakening of community structure. Then this process will repeat again and again. In healthy state, the very large w will separate S and I cluster completely and rapidly, thus the community structure is increasingly strong in the early stage and keeps $Q_n \approx 1$ after S and I clusters are separated (see Fig. 3(d)).

From the above, S nodes and I nodes can be divided into two loosely coupled but internally tight communities due to the rewiring mechanism in the transient process. One question is whether this property is helpful for the control of disease. In this viewpoint, we study the impacts of adaptive community structure on the control of disease, and pursue efficient control strategies. Look back to transient processes in the four phases of the system. In endemic state, disease prevails so rapidly that it is difficult to detect the community structure and control the epidemic; In healthy state, there is no need for controlling disease; In oscillatory state, the oscillatory phenomenon occurs only when $i(0)$ is very large, such as $i(0) > 0.9$, but it is somewhat unrealistic. In bistable state, small $i(0)$ can arouse the outbreak, and there is a very strong community structure for a long time. Therefore, we only focus on the bistable state.

In the transient process, the strength of community

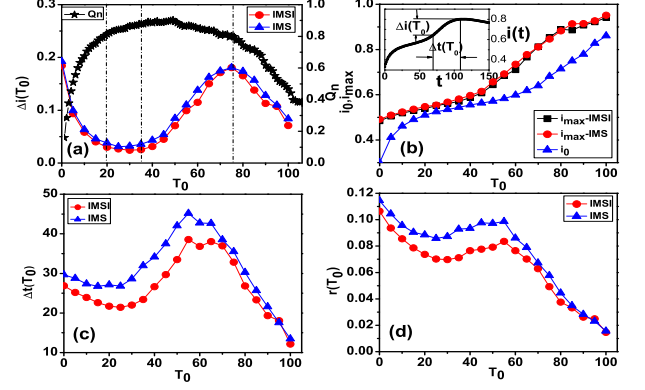


FIG. 3: (Color online) The effects of different immunization strategies vs the starting time of immunization T_0 . (a) $\Delta i(T_0)$, (b) i_0, i_{max} , (c) $\Delta t(T_0)$, (d) $r(T_0)$. Here $i(0) = 0.3, w = 0.3, r = 0.002, p = 0.008$, and $f = 0.008$. “Stars” in (a) represent the time evolution of $Q_n(t)$. Inset of (b) shows the definition of $\Delta i(T_0)$ and $\Delta t(T_0)$. Each point represents an average over 200 realizations.

structure is always changing. To understand the effects of adaptive community structures on the prevention of epidemic, we compare the control effects started from different stages. As we all know, immunization and quarantine are two basic measures to control epidemic spreading. Here we consider immunization strategy and quarantine strategy, respectively.

Immunization strategy—immunizing a fraction f of S nodes per time step. Two approaches to choose S nodes are compared: (1) randomly choose S nodes from the network (labelled IMR); (2) randomly select S nodes from SI links (labelled IMSI). To evaluate the effect of the control strategy, we let T_0 be the starting time of immunization (quarantine), $\Delta i(T_0)$ be the difference between the density of infected $i(T_0)$ at time T_0 and the maximal density of infected $i_{max}(T_0)$ that can reach after immunization (quarantine), i. e., $\Delta i(T_0) = i_{max}(T_0) - i(T_0)$, $\Delta t(T_0)$ denote the time interval of this process, and $r(T_0)$ denote the total percentage of immunized (quarantined) nodes in $\Delta t(T_0)$ (see inset of Fig. 3 (b)).

In Fig. 3, the results of IMR and IMSI strategies are compared. Figs. 3 (a), (c) and (d) show that these curves follow similar trends. What is more interesting is that three parameters are minimal when $T_0 \in [20, 35]$. So the optimal starting time of immunization is $T_0 \in [20, 35]$ which is somewhat against our intuition – the earlier immunization starts, the better control effect is. To explicitly explain such a phenomenon, the time window of T_0 is divided into four regions according to the trend of $\Delta i(T_0)$ (see Fig. 3 (a)): $\Delta i(T_0)$ decreases for $T_0 \in [0, 20]$, keeps stable for $T_0 \in [20, 35]$, increases gradually for $T_0 \in [35, 75]$ and drops again for $T_0 \in [75, 100]$.

For $T_0 \in [0, 20]$, the strength of community structure

and the density of infected $i(t)$ increases rapidly. When the community strength is weak, immunizing few S nodes can not effectively break the bridges between S cluster and I cluster. Thus, the increase of prevalence is large, e. g., $\Delta i(T_0 = 0) \approx 0.2$. As the delaying of the starting time T_0 , the community structure will be enhanced, and thus IMSI strategy can break the connections between the two communities more thoroughly, which makes the prevalence soon be brought under control. Consequently, $\Delta i(T_0)$ decreases as T_0 , which is exactly opposite to traditional concept “the earlier, the better”. Meanwhile, the epidemic can be inhibited faster than before (i.e., smaller $\Delta t(T_0)$ in Fig. 3(c)) with immunizing fewer S nodes on SI links (i.e., $r(T_0)$ in Fig. 3(d)).

For $T_0 \in [20, 35]$, the community structure is very strong and the density of infected $i(t)$ increases slowly. Owing to the very strong community structure, immunizing S nodes on SI links can timely hold back the re-outbreak in S cluster, which results in the minimum of $\Delta i(T_0)$. In Figs. 3 (c) and (d), $\Delta t(T_0)$ and $r(T_0)$ also reach their minimum and keep stable.

For $T_0 \in [35, 70]$, the adaptive network also has very strong community structure. However, it will certainly take some time to hold back the increase of prevalence, e. g., $\Delta t(T_0) \approx 20$ for $T_0 = 20$. Suppose immunization starts after $T_0 = 35$, there is not enough time left for controlling, because tight S cluster is invaded gradually by epidemic again which leads to the weakening of community structure at $t \approx 50$ (see Fig. 1(b)). So re-outbreak is inevitable due to the lack of enough controlling time. Therefore, the latter the starting immunization time T_0 , the worse the control effect of IMSI strategy.

For $T_0 \in [70, 100]$, the prevalence increases rapidly, while the strength of community structure decreases dramatically. Though the trends of these curves are the same as the region $T_0 \in [0, 20]$, the reason is completely different. $i_{max}(T_0) \rightarrow 1$ for $T_0 \in [70, 100]$ (see Fig. 3(b)), because control strategies no longer have any significance in this stage. $\Delta i(T_0) \approx 1 - i(T_0)$ thus decreases with T_0 .

Interestingly, in Fig. 3, the impact of IMS strategy is almost equal to IMSI strategy. To explain this reason, the percentage of S nodes on SI links in all S nodes (S_r/S) is shown in Fig. 4. Although there is the strongest community structure at $t \approx 50$, the ratio $S_r/S \approx 0.5$ is still relatively large. Consequently, both IMS strategy and IMSI strategy have similar results.

Quarantine strategy – quarantining a fraction f of I nodes per time step. Similar to immunization, two approaches to choose I nodes are also considered: (1) randomly choose I nodes from the network (labelled ISR); (2) randomly selected I nodes from SI links (labelled ISSI).

The effects of ISR strategy and ISSI strategy are compared in Fig. 5. Unlike the immunization strategies, the effect of ISR strategy is much worse than ISSI strategy. As illustrated in Fig. 4, the ratio I_r/I is small, that is, most of infected nodes are not on the SI links. As a result, ISR strategy can hardly pitch on the I nodes on SI

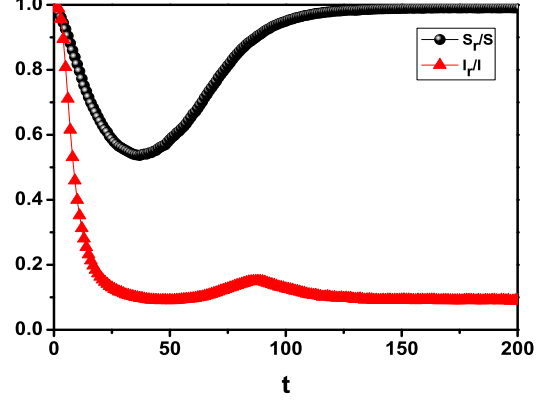


FIG. 4: (Color online) In bistable state, S_r/S and I_r/I vs time t , where S_r/S (“circles”) represents the percentage of S nodes located on SI links in all S nodes and I_r/I (“triangles”) represents the percentage of I nodes located on SI links in all I nodes. Results are derived from an average over 200 realizations.

links, and the advantage of community structure could not be well developed by ISR strategy. However, the effect of ISSI strategy is striking since ISSI strategy can efficiently cut the pathways of disease to the S cluster. Especially, $\Delta i(T_0)$ in Fig. 5 (a), $\Delta t(T_0)$ in Fig. 5 (c) and $r(T_0)$ in Fig. 5 (d) reach the minimum when $T_0 \in [20, 65]$, which corresponds to the time interval of strongest community structure (Here, we should note that, compared with IMSI strategy, ISSI strategy is more efficient in controlling the epidemic and has larger optimal region since ISSI strategy can directly cut more spreading pathways to the S cluster than IMSI strategy). The reasons of the trends of these curves in Fig. 5 are similar to the case of immunization strategy: In initial stage ($T_0 \in [0, 20]$), community strength increases with T_0 . Therefore, larger T_0 brings about better control effect. Then it reaches the optimal region, i. e., $T_0 \in [20, 65]$, in which the community structure is strongest. ISSI strategy can contain epidemic spreading rapidly. So $\Delta i(T_0)$, $\Delta t(T_0)$ and $r(T_0)$ reach the minimum and keep stable. When $T_0 \in [65, 90]$, the compact S cluster is invaded by disease again and the community structure becomes weak gradually, so the effect of ISSI strategy become poor with T_0 . At last, $i_{max}(T_0) \approx 1$ for $T_0 > 90$, thus $\Delta i(T_0)$ decreases with T_0 again.

To sum up, we studied the properties of community structures in the transient process of adaptive networks by the standardized modularity. We found that different degrees of community strength emerge from distinct rewiring conditions. Especially in bistable state, the very strong community structure can hold for a long period. In view of this, community-based immunization strategies and quarantine strategies are studied thoroughly. Because most S nodes are on SI links when epidemic pre-

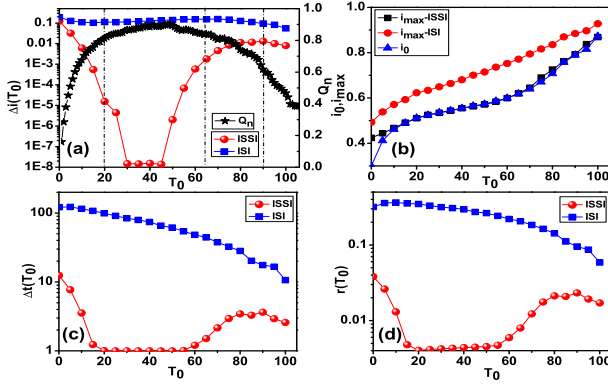


FIG. 5: (Color online) The effect of different quarantine strategies vs the starting time of quarantine T_0 . (a) $\Delta i(T_0)$, (b) i_0, i_{max} , (c) $\Delta t(T_0)$, (d) $r(T_0)$. Here $i(0) = 0.3, w = 0.3, r = 0.002, p = 0.008$, and $f = 0.008$. “Stars” in (a) represent the time evolution of $Q_n(t)$. Each point represents an average over 200 realizations.

vails in an adaptive network, random immunizing S nodes can give rise to similar effects as immunizing S nodes on SI links. Nevertheless, for quarantine strategy, quarantining I nodes on SI links is significantly better than random quarantining I nodes in the network, since the for-

mer can efficiently cut the pathways of epidemic invading to the S clusters. Both the study of immunization and quarantine strategies discover a counter-intuitive conclusion: it is not “the earlier, the better” for the implementing of control measures. And the optimal control effect is obtained if control measures can be efficiently implemented in the period of strong community structure. More significantly, community-based quarantine strategy plays more efficient performance than community-based immunization strategy.

The prevalence of an infectious disease can trigger behavioral responses of people trying to minimize the risk of being infected. If so, further study on the control strategy in adaptive networks has instructive significance. And we have done a forward step along this line. This is helpful for controlling adaptive dynamics in real world, such as epidemic and rumor spreading.

Acknowledgments

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